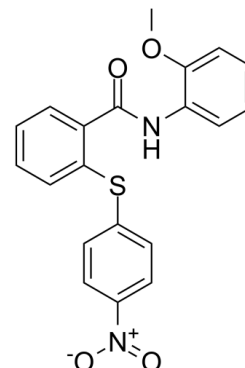


## Data Sheet

<b>Product Name:</b>	RN-18
<b>Cat. No.:</b>	CS-6953
<b>CAS No.:</b>	431980-38-0
<b>Molecular Formula:</b>	C <sub>20</sub> H <sub>16</sub> N <sub>2</sub> O <sub>4</sub> S
<b>Molecular Weight:</b>	380.42
<b>Target:</b>	HIV
<b>Pathway:</b>	Anti-infection
<b>Solubility:</b>	H <sub>2</sub> O : < 0.1 mg/mL (insoluble); DMSO : 100 mg/mL (262.87 mM); Need ultrasonic



### BIOLOGICAL ACTIVITY:

RN-18 is a HIV-1 viral infectivity factor (**HIV-1 Vif**) inhibitor with an **IC<sub>50</sub>** of 6  $\mu$ M in nonpermissive H9 cells. **IC<sub>50</sub> & Target:** IC<sub>50</sub>: 6  $\mu$ M (nonpermissive H9 cell)<sup>[1]</sup> **In Vitro:** RN-18 and RN-19 exhibits potent antiviral activity in the nonpermissive H9 and CEM cells but not in MT4 or CEM-SS cells, confirming that the antiviral activity was Vif specific. RN-18 shows the greater potency (IC<sub>50</sub>=4.5  $\mu$ M in CEM cells) and specificity (IC<sub>50</sub>>100  $\mu$ M in MT4 cells) among the two compounds<sup>[1]</sup>. In the presence of the inhibitor, RN-18, reverse transcriptase activity in the nonpermissive H9 and CEM cells decreases substantially and in a dose-dependent manner. RN-18 also exhibits antiviral activity in CEM-SS modified to stably express A3G but does not exhibit antiviral activity in the parental CEM-SS cell line. RN-18 antagonizes Vif function and inhibits HIV-1 replication only in the presence of A3G. RN-18 increases cellular A3G levels in a Vif-dependent manner and increases A3G incorporation into virions without inhibiting general proteasome-mediated protein degradation. RN-18 enhances Vif degradation only in the presence of A3G, reduces viral infectivity by increasing A3G incorporation into virions and enhances cytidine deamination of the viral genome<sup>[2]</sup>.

### PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay:** RN-18 is dissolved in 0.1% DMSO.<sup>[2]</sup> H9 or MT4 cells are treated overnight with 0, 1, 5, 10, 25 or 50  $\mu$ M RN-18 (all at 0.1% DMSO) and infected with HIV-1. All cells are maintained in the presence of DMSO or RN-18 for 14 d, and viral replication is monitored every 2 d by measuring reverse transcriptase activity in culture supernatants. The average % relative infectivity at day 7 is determined from 3 separate reverse transcriptase assays. Grafit software is used to fit curves and to determine IC<sub>50</sub><sup>[2]</sup>.

### References:

- [1]. Mohammed I, et al. SAR and Lead Optimization of an HIV-1 Vif-APOBEC3G Axis Inhibitor. ACS Med Chem Lett. 2012 Jun 14;3(6):465-469.  
[2]. Nathans R, et al. Small-molecule inhibition of HIV-1 Vif. Nat Biotechnol. 2008 Oct;26(10):1187-92.

### CAIndexNames:

Benzamide, N-(2-methoxyphenyl)-2-[(4-nitrophenyl)thio]-

### SMILES:

O=C(NC1=CC=CC=C1OC)C2=CC=CC=C2SC3=CC=C([N+](=O)[O-])C=C3

**Caution: Product has not been fully validated for medical applications. For research use only.**

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